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THE IMPORTANCE OF CLINICAL EXAMINATION FOR THE DIAGNOSIS OF HEART DISEASE AND LEFT CONGESTIVE HEART FAILURE SYNDROME IN DOGS AND CATS - A REVIEW

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Abstract

The aim of this systematic review is to present the clinical examination of the cardiovascular system in dogs and cats. The heart is an organ deeply interconnected with the hemodynamic of the entire body. Anamnesis and physical examination allow early diagnosis of heart disease. The stages of the examination are represented by inspection, palpation, percussion, auscultation and thermometry. This can establish the treatment schedule, prolonging the patient's life and improving its quality. Important clinical changes in left congestive heart failure syndrome must be quickly and correctly identified: respiratory distress, cough, syncope, heart murmur, or gallop sound. This allows the establishment of emergency therapeutic protocol. Clinical monitoring is still important for both the clinician and the owner. It shows the effectiveness of the action of drugs and the risk of cardiovascular decompensation, requiring adaptation of therapy.

Key words*: clinical, cardiovascular, dyspnea, murmur, heart failure.*

INTRODUCTION

Despite the increasing diversity and complexity of cardiac diagnostic examinations, the information obtained from the medical history and clinical examination have undeniable value. History and clinical examination offer diagnostic clues and guide the initial therapy in emergency situations involving cardiac, respiratory and vascular diseases. This information can allow the diagnosis of cardiac pathology, help to differentiate or associate the condition to respiratory pathology, prioritize diagnostic tests, and provide objective evidence of a therapeutic response.

MEDICAL HISTORY

In the first stage, identifies age, sex and breed (Keene et al., 2019), sometimes referred to as the "signalment". The history (anamnesis) should include key questions that include: (1) the reason for the consultation; (2) the onset of signs; (3) duration of the medical problem; (4) the evolution of signs; (5) the vaccination and heartworm prophylaxis history; (6) current medications and the animal's response to therapy; and finally, (7) the owner's ability to administer the drugs. Additional questions that further define the major problems should also be directed to the owner. For example, useful information about respiratory difficulties might include its relationship to rest or exertion; an onset that is sudden or gradual; and any association with audible respiratory noise that might indicate a major airway obstruction.

Important information highlighted in the medical history report is particularly relevant to the pathology of the cardiorespiratory system. Increased respiratory rate (tachypnea), respiratory distress (hyperpnea or dyspnea), coughing, exercise intolerance, difficulty in sleeping, and general signs of illness are common to both cardiac and respiratory disorders. Abnormal respiratory patterns are usually overlooked by owner and are discussed further under "Clinical Examination".

Further information to determine from the history or direct observation includes: (1) anorexia or decreased appetite; (2) weight loss or cachexia; (3) abdominal distention (hepatomegaly or ascites); (4) diarrhea (from severe right heart failure with intestinal edema and potentially enteropathy with protein loss);

(5) the presence of hemoglobinuria (found in caval syndrome of dirofilariosis); (6) hemoptysis (in pulmonary thromboembolism, pneumonia, coagulation disorders including toxicities, foreign bodies, and neoplasia); (7) exertional collapse, syncope or seizures; and (8) signs of hind leg instability or sudden onset of paresis/paralysis (Smith et al., 2015).

Syncope can be to cardiac, vascular, or respiratory origin. When due to respiratory disorders exertional collapse or syncope is often associated with pulmonary arterial hypertension, severe bronchopulmonary or pleural space diseases, or obstructive upper respiratory diseases (especially in brachycephalic breeds). While less common, syncope in respiratory disease can develop from low arterial oxygen concentration, insufficient cardiac output (pulmonary arterial hypertension), or potentially from activation of reflex bradycardia as with vasovagal syncope (Kittleson, 1998).

Cardiogenic syncope has many causes and some of these might become more relevant based on the history and clinical examination. For example, a cardiogenic mechanism is suggested by signs of reduced cardiac output as in a dog with dilated cardiomyopathy or one with severe mitral regurgitation. Reflex-mediated (vasovagal) syncope can occur in dogs with structural heart disease, especially with sudden excitement or exertion. Cardiac brady- and tachyarrhythmias are a common reason for syncope and might be detected during clinical examination. Examples include sinus arrest, complete atrioventricular block, or sustained ventricular tachycardia. Abnormal auscultation might suggest pulmonary hypertension or a congenital cardiac defect associated with syncope such as subaortic stenosis, pulmonary stenosis, or tetralogy of Fallot (Tilley et al., 2008). Often there are multiple factors, as with ventricular arrhythmia in a dog with dilated cardiomyopathy.

Syncope with a cardiogenic origin is associated with sudden loss of consciousness, short duration (usually less than one minute), and rapid recovery. The syncope is often induced by exercise or excitement/stress. Sometimes the event is preceded by vocalization. Pre- and postictal behavioral abnormalities are absent. Urination can occur although loss of feces is uncommon. Tonic-clonic muscle contractions, facial fits, and hypersalivation are not typical of cardiac syncope. These aspects help to differentiate canine syncope from seizure disorders. Syncope in cats is more challenging to assess and often shares features of neurological disease (Penning V.A., 2009). One useful recommendation is to videorecord the animal during the crisis as it can provide valuable clues which complete the history and physical examination.

Exertional weakness and especially **intolerance to exercise** are sensitive though not specific signs of cardiac disease. Often, the owners do not report exercise intolerance. In some cases, the dog is relatively sedentary, as with many English Bulldogs (Chong, 2017). Cats sleep almost all the time with only short periods of high activity so owners might not identify their clinical signs until they become severe. Furthermore, dogs are excellent athletes and are not commonly stressed to the maximal exercise capacity that might reveal underlying heart disease.

Moreover, these findings with exercise are nonspecific, and can develop with respiratory, musculoskeletal, neurologic, and some systemic diseases. Other conditions that might show intolerance to effort can include anemia, endocrinologic (adrenal diseases, thyroid diseases, diabetes mellitus), and some metabolic diseases (including hypokalemia).

Coughing is probably the most common symptom reported by owners of dogs with heart disease. However, frequently the cough is not due to cardiac disease. Potential etiologies of a "cardiac" cough in dogs include alveolar pulmonary edema and marked compression of the left principal bronchus between the left atrium and the aorta. The importance of bronchial compression is debated, but most studies have either focused on patients undergoing bronchoscopy with a high likelihood of concurrent bronchitis and bronchomalacia, or else relied only on radiographs to exclude bronchitis (which is not sufficient). Clearly many dogs with chronic valve disease also have concurrent tracheal disease, chronic bronchitis, or pulmonary fibrosis which can result in coughing or tachypnea in the absence of cardiac enlargement or failure.

In terms of specific causes of coughing, the following are general guidelines. Canine patients with upper respiratory diseases usually exhibit a chronic, loud cough with high acoustic intensity, paroxysmal bouts, and worsened by exertion of (Martin, 1997). The "cardiac" cough often described as low-intensity and occurs intermittently, being favored by the presence of compression of the main left bronchi by the dilated left atrium. It can be accompanied by dyspnea and nocturnal agitation (Ferasin, 2019). As indicated above, many of these dogs have concurrent airway diseases, including bronchomalacia, that make the source of the cough uncertain. For dogs developing left congestive heart failure any coughing can be associated with tachypnea and possibly dyspnea. In fulminating failure there might be bloody or foamy nasal secretions. Generally, these dogs have acute respiratory effort.

The abnormalities mentioned above are important considerations when evaluating a potential case of cardiac or respiratory disease. Frequently, a complete and correctly highlighted history will guide the veterinarian to the nature of the animal's underlying pathology, often allowing the differentiation of heart disease from other competing problems (Rijnberk, 1995).

GENERAL EXAMINATION

The general appearance of the canine or feline patient, as well as the overall condition of maintenance are observed. The attitude, posture, and behavior of the animal provide clues about the type of pathology and its severity. Vital signs and rapid thoracic auscultation also provide valuable clues. Animals with respiratory distress often require immediate oxygen supplementation and sedation to reduce symptoms and permit a more thorough examination.

General Inspection

Weight loss occurs in chronic, severe heart disease. It is represented by the loss of fat and skeletal muscle mass, despite the maintenance of appetite (Ineson, 2019).

Obesity can restrict ventilation and might precipitate coughing in cases of tracheal collapse. Also, obesity exacerbates the cough associated with lung or heart disease (Slupe et al., 2008).

Posture of an animal can be revealing and often reveals signs of respiratory dysfunction. One that refuses to adopt a decubitus position may have respiratory distress due to pulmonary edema, pleural effusion, pericardial effusion, pneumothorax, diaphragmatic hernia, or another respiratory condition. Dogs often assume a standing or sitting position to minimize their work of breathing; cats typically position in sternal recumbency with abducted elbows. The patient with head in extension, abducted elbows, open mouth breathing, and dilated (flared) nostrils, requires immediate therapy for respiratory distress (Le Boedec et al., 2012). Orthopnea is discussed more fully below.

Vital signs and Thermography

The pulse and respiratory rates, body temperature, and a rapid assessment of mucous membrane color and refill time should be obtained from any patient as part of the initial evaluation (assessment of membranes is discussed later). Ideally, a noninvasive systemic arterial blood pressure also should be also recorded.

Significant changes in body temperature results from a multitude of etiological factors, including changes in metabolic rate, impairment of tissue perfusion, toxic factors, exposure to extreme environmental temperatures, inflammatory or infectious diseases and iatrogenic issues. From a cardiovascular perspective, hypothermia can be induced by the presence of hypotension, bradyarrhythmia or tachycardia (atrial fibrillation, tachycardia or ventricular fibrillation) or cardiogenic shock. Fever or hyperthermia can be associated with infective endocarditis, infection or inflammation of the myocardium or pericardium, or represent increased temperature related to work of breathing, as with upper airway pathology found in brachycephalic dog breeds (Pope, 2009).

A true fever must be distinguished from hyperthermia secondary to anxiety or increased work of breathing. Hyperthermia usually resolves once a dyspneic patient is sedated and the underlying problem is managed. Hypothermia – especially in cats – is often associated systemic arterial thromboembolism or shock. Cardiogenic shock is associated with bradycardia and systemic hypotension.

Initial Auscultation

In patients with signs of respiratory dysfunction, a rapid auscultation assessment of the lungs might reveal important abnormalities such as muffled breath and heart sounds (suggesting a pleural or pericardial fluid accumulation), lung crackles (indicating parenchymal or small airway dysfunction), or sounds of inspiratory (upper airway) or expiratory (lower airway) airway obstruction. Lung ultrasound scanning is complementary to thoracic auscultation. Similarly, a rapid cardiac auscultation can be revealing. For example, the presence of marked sinus arrhythmia along with coughing suggests a primary respiratory problem, even in the presence of the left apical murmur. Conversely, sinus tachycardia with a loud murmur, gallop sounds, or obvious arrhythmia such as atrial fibrillation points to a cardiac cause of respiratory dysfunction. Notably, cardiac findings are often absent in many cats with congestive heart failure, at least during initial examination. More details about auscultation follow under "Cardiorespiratory Examination".

Initial Diagnostic Tests

Diagnostic testing must be performed with great care in patients with symptomatic respiratory or heart diseases. One must avoid aggressive manipulation or performing lower-priority tests (for example, radiography or detailed cardiac ultrasound) (Vulpe et al., 2014). In addition to the initial inspection, recording of vital signs, and rapid auscultation, point-of-care ultrasound should be given priority using a standardized protocol for the practice.

CARDIORESPIRATORY EXAMINATION

The specific clinical examination requires following a rigorous protocol, taking into account that each stage of evaluation can provide valuable information for obtaining a diagnosis. These stages include inspection, palpation, percussion, and auscultation of the heart and lungs.

Evaluation of visible mucous membranes

It is recommended to assess mucous membrane color both at the level of the cephalic extremity (oral mucosa) and caudal (preputial or vaginal mucosa). Assessment of capillary refill time will also be considered. A refill time of more than 2 seconds suggests peripheral vasoconstriction, typically as a response to decreased cardiac output. Pale mucous membranes suggest a diminished cardiac output, shock, or anemia (Ware et al., 2021).

The hyperemic appearance of mucous membranes (plethora) may indicate venous congestion (in right congestive heart failure) or polycythemia (in right-left congenital heart shunts, encountered within the septal ventricular defect or persistence of the arterial duct). Peripheral vasodilation from sepsis or vasodilator drugs are other causes.

Cyanosis is classified as central (low arterial oxygen content) or peripheral (due to intense vasoconstriction from hypotension or reduced perfusion). In most cases cyanosis indicates a low oxygen tension, either centrally or due to obstruction of arterial blood flow. For example, lack of oxygen diffusion at the alveolar level or intrapulmonary shunting can accompany respiratory disease and distress. Similarly, a low arterial oxygen can be due to right-to-left shunting defects at the level of the heart, as with tetralogy of Fallot.

Differential cyanosis affecting the caudal part of the body could indicate a right-to-left ("reversed") patient ductus arteriosus with severe pulmonary hypertension. A similar finding can occur in cat with distal aortic thromboembolism where there is the pale or cyanotic appearance of the affected, ischemic limbs (Smith et al., 2004).

Usually, cyanosis is a late sign in acquired cardiac disease associated with pulmonary dysfunction due to lung edema or atelectasis from pleural effusion. It can also present as a symptom of hypoxemia in dogs with primary or chronic obstructive respiratory diseases.

The assessment of respiratory rate and pattern of ventilation

Respiratory pattern and respiratory rate often change with heart disease: the degree of tachypnea and dyspnea usually reflect the severity of the heart pathology (Dickson, 2018). Normal *respiratory rate* in dogs and cats is less than 30 respiration per minute (rpm). Respiratory frequency exceeding 30 rpm is

considered to be tachypnea (Ohad, 2013; Boswood, 2020). Often but variably changes in respiratory rate or pattern is associated with altered breath sounds detectable during respiratory auscultation (also see Auscultation later).

Tachypnea (increased respiratory rate without distress or increased depth of ventilation) has numerous causes, including lung and pleural space disorders that can arise from congestive heart failure. Tachypnea minimizes the work of breathing when the lung is restricted such that it represents an early sign of pulmonary edema or pleural effusion that might be recognized by the observant client during home monitoring. It is important not to confuse simple tachypnea, or polypnea, due to thermoregulation (in dogs), stress, fever, or pain with tachypnea caused by cardiac, respiratory or systemic pathology or intoxications. This distinction often requires further diagnostic testing such as thoracic ultrasound or radiography.

Dyspnea is the sensation of difficult, sometimes painful, breathing reported by people. In veterinary medicine it is sometimes used to indicate respiratory distress or *hyperpnea* (increased rate and depth of ventilation). Regardless of the terminology used, dyspnea is an important sign of respiratory, cardiac, or systemic disease and requires rapid etiological diagnosis to institute life-saving therapy.

The causes of respiratory distress or dyspnea are numerous and can be tracked from the upper airways, through the bronchopulmonary system, to the pleural space and even the muscles of ventilation. Animals with extra thoracic airway obstruction have a pronounced or more rapid respiratory effort. (Corcoran, 2010) as do dogs with severe abdominal distension from any cause.

Respiratory rate is not significantly increased as long inspiration usually reduces the work of breathing, and exhalation is usually normal (MacPhail, 2014). However, if the dog develops hyperthermia, both increased depth and rate might be detected. Patients with fixed upper airway obstructions tend to have prolonged phases of both inhalation and exhalation.

The most common cardiac cause of dyspnea in dogs is pulmonary edema following left-sided congestive heart failure. In cats, dyspnea of cardiac origin often indicates the presence of pleural effusion and/or pulmonary edema, also associated with congestive heart failure. Edema might be associated with abnormal auscultation, variably louder bronchial sounds or crackles. Pleural effusions result in louder referred tracheal sounds dorsally with muffling of breath and heart sound ventrally (fluid line).

More severe respiratory distress is clinically translated by the presence of discordant or paradoxical pattern of breathing, evidenced by the presence of movement of the chest and abdominal walls inward, due to the diaphragm contraction during inspiration (Little, 2012). It is called paradoxical because it opposes the normal expansion of the thoracic cavity, thus aggravating respiratory failure. The observation of this respiratory type draws attention to the presence of a severe pathology and requires rapid therapeutic action (Cole, 2008).

Progressive, chronic tachypnea or dyspnea also may be associated with right-sided congestive heart failure secondary to ascites or pleural effusion. These signs are also observed in some dogs with cardiac compression due to large pericardial effusions.

Orthopnea in humans indicates an inability to breath comfortably while recumbent. As mentioned under “General Examination” dogs and cats with respiratory distress assume certain breathing positions. Typically, there is stretching and elevation of the neck and head and abducted elbows to open the thoracic inlet and expand the chest cavity. Dogs typically stand or sit; cats assume sternal recumbency. The presence of a frightened facies with flared nostrils and retracted lips often indicates serious pulmonary dysfunction or pleural effusion. These animals have minimal respiratory reserve and the least stress can be fatal (Sigrist, 2011).

Animals alter their pattern of ventilation to minimize the work of breathing. Patients with dynamic upper airway obstructions, such as laryngeal paralysis, can develop respiratory distress that is often worsened by exercise. Aditionally, these animals have prolonged inspiration, as negative intrathoracic pressure tends to collapse the affected area, narrowing the lumen. Obstructive inspiratory sounds are detectable with the stethoscope and frequently audible during observation alone.

Animals with intrathoracic airway obstructions, such as dogs with bronchomalacia or chronic bronchitis or cats with asthma, tend to develop increased respiratory effort during exhalation. Bronchoconstriction usually creates a whistling or high pitched sound (wheeze) during expiration association with contraction of the abdominal muscles that improve the expulsion of air from the lungs (Corcoran, 1995).

Examination of the Jugular Veins

Distention of jugular veins and jugular pulsation extending towards the mandible are clinical signs of right-heart dysfunction. The examination is performed with the animal in a standing position and the head elevated parallel with jaw parallel to the floor. Abnormal pulsations are associated with elevated central venous pressure, vigorous right atrial contraction, tricuspid regurgitation, and arrhythmias causing atrioventricular dissociation. Jugular pulsations might be accentuated during abdominal compression (abdomino- or hepto-jugular reflex).

The jugular pulse identified with right-sided congestive heart failure is often related to tricuspid regurgitation that increases right atrial pressure during systole. In congenital pulmonary stenosis or with pulmonary hypertension the pulse occurs when the right atrium contracts more vigorously in end-diastole against a less compliant, hypertrophied ventricle (Radulescu, 2019). With ventricular tachycardia or complete atrioventricular block the jugular pulse is intermittent associated with atrial contraction on a closed tricuspid valve (cannon waves)

Distention of jugular veins indicates increased systemic venous pressure or obstruction to venous return. It occurs in right heart failure, pericardial effusions, heart base tumors and with large mediastinal masses. It can also be seen with over-infusion of intravenous fluids, especially in cats with otherwise mild cardiac dysfunction or those in volume-retentive states (Ionita, 2000).

Palpation

The cervical region of cats (and dogs) should also be evaluated for the presence of lymphadenopathy and thyroid tumors that in cats are often associated with secondary cardiac changes. In dogs thyroid carcinoma can partially obstruct the airway and often metastasizes to the thorax.

Palpation of the trachea can highlight a collapse, tumor masses or increased sensitivity. A slight pressure exerted from the side inwards of the larynx can exacerbate the inspiratory stridor in dogs with laryngeal paralysis (Ware, 2011).

Palpation of the chest is necessary to identify the point of maximum intensity (PMI) of the heart (apical) beats and recognize precordial vibrations (thrills) of a loud cardiac murmur. The normal cardiac impulse (apical beat) is located at the left side, in the 4^{th} to 6^{th} intercostal spaces, usually at the 5^{th} ICS in dogs. Decrease in intensity can occur with obesity, pleural or pericardial effusions, an intrathoracic mass, pneumothorax, or in cases of depressed cardiac contractility. Increased apical beat intensity occurs in young or thin animals or in setting of a hyperdynamic circulation. Left ventricular dilation can displace the ventricular apex ventrally and caudally. Right ventricular hypertrophy can increase the intensity of the right apical impulse (normally at the 3rd or 4^{th} intercostal space and weaker than the left apical impulse).

Palpation of the abdomen can identify hepatomegaly and ascites in right-sided congestive heart failure. Other abnormalities, such as tumor masses or lymphadenopathy might be found suggesting the presence of lung metastases.

Abdominal palpation to identify ascites or hepato- or splenomegaly is difficult to achieve in obese animals (ultrasound investigation is preferred). In cats, palpation of the kidneys with small dimensions usually indicates chronic kidney disease and can be associated with systemic hypertension (Stepien, 2011). Irregular renal surfaces representing prior infarctions are sometimes identified in cats with cardiomyopathy.

The arterial pulse is palpated and analyzed in standing position, most frequently at the femoral artery. It is analyzed in terms of rate, regularity (rhythm), quality (intensity) and symmetry. In congestive heart failure, tachycardia is noted, translated by an increase in pulse frequency, while in respiratory diseases it is more likely to notice a normal rate of it, or the presence of sinus arrhythmia

(Smith et al., 2015). Pulse deficits are an important indicator of a cardiac arrhythmia (as in atrial fibrillation).

The hypokinetic pulse is characteristic of diseases with low stroke volume, as with heart failure, or impaired ejection dynamics, as with subaortic stenosis. The hyperkinetic (bounding) pulse indicates the presence of a widened pulse pressure between systole (normal to increased) and diastole (lower than normal). Typical causes are left-to-right patent ductus arteriosus, moderate to severe aortic regurgitation, and third degree atrioventricular block with ventricular escape rhythm.

Percussion

Chest percussion is a very efficient method for recognition of large pleural effusions but requires experience and is a lost clinical art. The typical features of pleural effusion are hyporesonant (duller) sounds bilaterally and the presence of a dorsal fluid line. Similar findings can occur with large mediastinal masses (cranially) or pulmonary masses. The area of cardiac dullness is also expanded with large pericardial effusions. Conversely hyperresonance on thoracic wall percussion may indicate the diagnosis of pneumothorax.

Auscultation

Auscultation is the most important stage of the clinical examination for detecting heart disease. Two fundamental abnormalities are identified by auscultation. The first involves the transient (brief) sounds, including the heart sounds. Abnormalities in the number, rate, rhythm, intensity, or character of the transient sounds might be detected. The second major abnormality detected by auscultation is the presence of pathologic cardiac murmur. Some key points regarding cardiac and respiratory auscultation are summarized below.

Technique Proper use of the stethoscope offers valuable clues for detecting cardiac and respiratory diseases. It must be done systematically and with care, with the animal in standing position, so that the heart is anatomically positioned. It is aimed at examining the heart, lungs and pleural space, and the upper respiratory tract (Dennis, 2013).

Auscultation should be done in a quiet environment. The examiner should listen carefully over the four canine heart valves, and over the left craniodorsal base of the heart (over the ascending aorta and pulmonary trunk). These areas should also be palpated for precordial thrills. In cats, auscultation is focused to the sternal edges both apically – near the palpable beat – and cranially (Fox, 1999).

For small dogs and cats it is recommended to use the pediatric stethoscope, mainly the diaphragm, because heart sounds of animals are best detected with this chest piece. Lower frequency sounds, especially the ventricular gallop in dogs and soft murmurs of aortic regurgitation are sometimes heard better with the stethoscope bell.

The left apical beat is initially identified by palpation. It represents the ventral part of the listening area for the mitral valve and corresponds to the intercostal space (ICS) 5 in most dogs, ventral to the costochondral junction. Mitral sounds project ventrally down the solid structure of the left ventricle towards the apex (Murmurs from the mitral valve are often loud at the apex as well as dorsal the mitral valve itself). From here, the stethoscope moves one intercostal cranially and dorsally to the aortic valve area (ICS 4); ventral and cranial to the aortic valve is the pulmonary valve (ICS 2-3). Murmurs from both semilunar valves project dorsally above the costochondral junction (Strickland et al., 2008).

Special attention is required to auscultation of the left axillary region, especially in young animals, for the detection of the continuous murmur of PDA.

Heart Sounds. The systolic heart sound S1 (generated by the vibrations around closure of the atrioventricular valves) is loudest over the mitral valve and left apical region. The second sound (indicating the onset of diastole) is generated by vibrations around the closure of the semilunar valves); it is most prominent over the aortic and pulmonary valve areas.

The intensity of the cardiac sounds may be diminished, suggesting the presence of pleural, pericardial effusion, tumor masses or pneumothorax. Depressed contractility from dilated cardiomyopathy is an under-recognized cause of soft heart sounds. The intensity of the first sound increases with progressive

cardiomegaly in primary (degenerative) mitral valve disease (Häggström, J., 1995).
Increased heart rate (tachycardia) is most often due to sympathetic activation. It can be seen as a physiological response (exertion, fear) or secondary to disease (fever, pain, anemia, hypovolemia, or heart failure).
Normal values for heart rate (Ettinger et al., 2017):

- Dogs: 70-160 (adult dogs); 60-140 (giant breeds); 80-180 (toy breeds); up to 220 in puppies;
- Cats: 140-240 (hospital measurement); 100-120 (home environment).

A decrease of the heart rate (bradycardia) is physiologically found in sighthounds (such as greyhounds), working athletic breeds (such as the border collie), and in the context of various organ or systemic pathologies, including hypothyroidism, hyperkalemia, hypothermia, and acute renal failure. Iatrogenic causes include sedatives and tranquilizers, beta-adrenergic blockers, and some antiarrhythmic drugs (Abbott, 2001) (Smith et al., 2008).
Cardiac rhythm disturbances might be recognized by auscultation. Dogs normally have sinus arrhythmia, typically associated with respiration. Exercise will usually resolve this, at least briefly. Exaggerated sinus arrhythmia might be noted in the setting of respiratory diseases; conversely, it is usually absent in heart failure (Bonagura et al., 1999). Pathologic rhythms can be intermittent and challenging to identify. For example, the pause after a single premature beat can mimic a sinus block or a sinus arrest. The same thing does not happen in the case of atrial fibrillation, which can be easily recognized by the presence of heart beats of different intensity accompanied by a noncyclical, chaotic rhythm.
Abnormal transient sounds usually indicate pathology. Splitting S1 or S2 sounds in dogs is usually caused by a conduction delay as with bundle branch block or ventricular ectopy. Splitting of S2 is mainly due to delayed closure of the pulmonary valve, which can also occur in pulmonary stenosis, ventricular septal defect or severe pulmonary hypertension.
An S3 (ventricular filling) or S4 (atrial contraction) sound is considered pathologic in dogs and in cats. These “gallops” are indicative of diastolic dysfunction, most often some form of cardiomyopathy or of congestive heart failure (Vancheri, 1989). Systolic clicks or additional sounds are generally due to valvular disease. The systolic click is most often attributed to the presence of mitral valve prolapse (de Madron, 2000). In most cases, isolated mitral and tricuspid valve systolic clicks are signs of mild valvular disease. However, gallops and systolic clicks in younger cats are especially concerning because they often indicate hypertrophic cardiomyopathy.
important indication for the presence of heart disease (Saponaro, 2023)

Cardiac murmurs are generated by the vibration of the cardiac anatomical structures under the influence of the blood flow that generates turbulence under certain conditions. Most often a murmur is associated with an increased ejection velocity (as with functional or physiologic murmurs) or a high velocity turbulent jet (as with most pathologic murmurs). High velocity jets are associated with valvular regurgitation and stenosis, restrictive ventricular septal defects, and left-to-right shunting PDA. However, there are other reasons for murmurs, including reduced blood viscosity (anemia), ejection into dilated great vessels, and sudden changes in the diameter of a flow pathway including fixed and dynamic obstructions (Côté et al., 2015).
The timing of the murmur in the heart cycle classifies it as systolic, diastolic, or continuous. Systolic murmurs are most common and occur during ventricular contraction. These include mitral and tricuspid regurgitation, ventricular septal defect, increased pulmonary flow of atrial septal defect, (sub)aortic and pulmonic stenosis, and those physiologic/innocent/functional murmurs not associated with cardiac disease. Timing can be subdivided into early (proto-), middle (meso-) or late (tele-) systole. Loud murmurs of ventricular septal defect and of mitral and tricuspid regurgitation (from myxomatous disease) are usually holosystolic. Some distinguish between holosystolic, where the second heart sound is still heard, and pansystolic where only the cardiac murmur is audible (for example - ventricular septal defect); however, this is less common today.

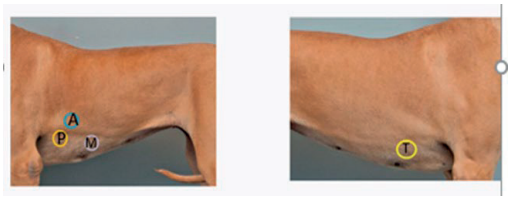
Diastolic murmurs are heard after S2; these are rare to uncommon in small animals. The most common example is the decrescendo diastolic murmur of aortic regurgitation caused by infective (bacterial) endocarditis (Smith et al., 2015). Stenosis of the mitral or tricuspid valve or inlets are rare.

Continuous murmurs are present throughout systole and diastole, as happens with persistence of the arterial duct (PDA). In this congenital heart defect, there persistent difference in pressure in between the aorta and the pulmonary artery generates a continuous murmur. Thus, one can hear a higher intensity murmur in systole (when the pressure differences are greatest) and a lower intensity murmur in the diastole. Should pulmonary hypertension develop, the diastolic murmur becomes softer or inaudible; this is often the case in cats.

The combination of systolic and diastolic murmurs can mimic a continuous murmur. In dogs this situation is most common with subaortic stenosis with moderate to severe aortic regurgitation, ventricular septal defect with secondary aortic insufficiency, and pulmonary stenosis accompanied by severe pulmonary regurgitation (or with pulmonary hypertension).

Phonocardiography – the graphical recording of heart sounds and murmurs – can specify the timing and the "shape" of a heart murmur, although this is not routinely done. For example, the crescendo-decrescendo (diamond-shaped) murmur of pulmonary or aortic stenosis contrasts with the more constant intensity ("plateau"-shaped) murmurs of mitral regurgitation and ventricular septal defect. This distinction is difficult to appreciate when listening with the stethoscope (Fonfara, 2015), especially when murmurs are loud.

The *point of maximal intensity* (PMI) of a murmur and its timing in the cardiac cycle are the most important clinical features distinguishing heart murmurs. Firstly, the typical PMI for different murmurs are over the respective valve areas.



A B

Figure 1. Representation of cardiac ascultation areas for the left (A) and right hemithorax (B) (after Englar R.E., 2017, modified)
(M - mitral valve area auscultation; A - aortic valve area auscultation; P - pulmonary valve area auscultation; T - tricuspid valve area auscultation)

Additionally, mitral regurgitation is heard at the left apex and radiates dorsally (and to the right if loud). Tricuspid regurgitation is over the right thorax (3-4 ICS) above the sternum, whereas, a typical murmur of VSD is loudest over the right sternal border (Englar R.E., 2017). Murmurs of (sub)aortic and pulmonic stenoses radiate cranially and for PS dorsally into the main pulmonary artery at the left-craniodorsal cardiac base. Subaortic stenosis also radiates towards the apex and into the right thorax (ascending aorta) when loud (Allen et al., 1998).

The murmur of PDA is also heard best over the left craniodorsal cardiac base because the high-velocity jet enters the pulmonary artery. Functional (physiologic) murmurs in dogs are nearly always soft, proto-mesosystolic in timing, and loudest over the aortic valve, pulmonic valve, or left craniodorsal cardiac base. In cats these functional murmurs are soft to moderate and typically due to sympathetic activation. The PMI in cats is over the left or the right cranial sternal edges.

Murmur intensity is most often classified into six grades, using a modified Levine grading scale:

- Grade 1 – faint, perceived after a few minutes of listening in a very quiet room. These are usually localized murmurs.
- Grade 2 – soft but immediately perceptible; usually focal, at the level of a single valve or listening area.

- Grade 3 – moderate intensity murmur that radiates to other adjacent listening areas.
- Grade 4 – loud, but not accompanied by a precordial vibration or thrill. Radiates widely.
- Grade 5 – loud murmur with a precordial thrill.

  Grade 6 – loud murmur audible without a stethoscope in some cases or with the chest piece off the thorax; always accompanied by a precordial thrill.

Numerous modifications of murmur grading appear in the veterinary literature with none following the exact original description of Levine (Cote, 2015). Some suggestions relate loudness of a murmur to intensity of heart sounds; others have condensed the scale to five or four grades of murmurs (Rishniw, 2018). There is no veterinary consensus at this point.

The murmur intensity does not always correlate with the severity of the disease. In some heart conditions there is this direct correlation, for example, in aortic stenosis and pulmonary stenosis the intensity and time of peak do correlate – louder and later are worse. In primary mitral valve disease of dogs, soft murmurs indicate mild disease and murmurs with precordial thrills have a greater likelihood for cardiac remodeling, but the "middle grades" have much overlap in terms of severity. In other heart diseases, the intensity of the murmur is not directly proportional to the severity: ventricular septal defect, mitral regurgitation associated with dilated cardiomyopathy, or murmurs in feline cardiomyopathy are good examples that correlate poorly (Visser, 2018).

Respiratory auscultation is performed at the level of the specific areas of each segment.

Normal breath sounds include tracheal sounds (from panting) in dogs, and bronchovesicular sounds over the thorax. There are both inspiratory and expiratory components to these sounds. Tracheal sounds are loudest over the cervical region and have a distinct "pause" between the phases. These usually radiate to the thorax where they are misinterpreted as pathologic "harsh" sounds (a description of no medical relevance). Tracheal sounds can help to exclude pleural effusions if well-heard from dorsal to ventral, bilaterally. Otherwise they are a nuisance that obscure other breath sounds.

Bronchovesicular sounds include contributions from both the lungs (vesicular) and larger airways (bronchial). There is little separation between phases with normal breathing. With respiratory disease, the sounds are often more intense during expiration. Accentuation is generally not a sign of high specificity for the presence of respiratory diseases. Asymmetry of bronchial sounds can develop if different lung lobes are affected; sometimes sounds become louder in diseased lungs but at other times they are attenuated, especially if there is bronchial obstruction. Attenuation of bronchovesicular sounds is also associated with obesity, atelectasis, pleural fluid, or thoracic masses. Adventitious respiratory sounds include stridor, wheezes, rhonchi, crackles (rales) and friction rubs. Critical upper airways obstructions are usually audible without the need for a stethoscope.

The perception of an increased sound or noise in upper airway auscultation (over the larynx and cervical trachea) suggests an obstructive upper respiratory condition. Respiratory *stridor* has high intensity, and accompanies the turbulent passage of air to the larynx or upper bronchial airways. It can occur both in fixed obstructions (it is evident in inspiration and exhalation) and in dynamic ones located at the upper airways (it appears only during inspiration).

Respiratory *stertor* is a low-toned noise, similar to snoring produced in the nasopharyngeal or pharyngeal pathway, generated by obstruction in one of these zones. It is most often due to soft palate redundancy and entrapment and frequently associated with stenotic phenomena of brachycephalic breeds. Increased nasal resistance usually accentuates the obstructive sounds. It is present during inspiration but might also be evident with exhalation (Dupré, 2016).

Abnormal respiratory sounds originating in the bronchial tree include *rhonchi* and *wheezes*. The rhonchus is similar in quality to stertor but localized to the thorax. It indicates fluid or mucous in larger bronchi. Wheezes are high-pitched expiratory sounds typical of bronchial narrowing as observed with feline bronchial asthma. Wheezes are also common when there is lobar or mainstem bronchial collapse or compression.

The detection of lung *crackles* (previously termed "rales") has distinct pathological relevance, being present in the inspiration and early expiration. In the setting of congestive heart failure it is a sign of severe pulmonary edema (Ettinger et al., 2016). However, the loudest crackles are frequently detected with pulmonary fibrosis or other primary lung disease that result in explosive opening of the smallest airways as this loose radial traction. Thus crackles cannot be assumed to be fluid.

CONCLUSIONS

Going through the stages of the clinical examination facilitates the establishment of the diagnosis of cardiac pathology, its differentiation or association with a respiratory pathology, as well as the evaluation of the proposed therapeutic response. Depending on the patient's condition, the emergency therapeutic conduct is instituted, or, if this allows, the paraclinical investigations with diagnostic value are continued.

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